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Effect of chronic alcohol consumption on plasma lipid, vitamins A, and E in Korean alcoholics

Jung Sook Seo[a,*], Kyung Mi Yang[b], Jung Mi Kim[c], Hyesun Min[d], Chang Su Kim[e], Betty J. Burri[f]

[a]*Department of Food and Nutrition, Yeungnam University, Gyeongsan 712-749, Korea*
[b]*Department of Cooking and Nutrition, Daegu Hanny University, Gyeongsan 712-715, Korea*
[c]*Department of Food and Nutrition, Daegu Science College, Daegu 702-723, Korea*
[d]*Department of Food and Nutrition, Hannam University, Daejon 306-791, Korea*
[e]*Department of Psychiatry, Daegu Mental Hospital, Daegu 711-830, Korea*
[f]*Western Human Nutrition Research Center, Agriculture Research Service, USDA, Davis, CA 95616, USA*

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Abstract

This human study was conducted to evaluate the effect of chronic alcohol consumption on plasma concentrations of lipid and the antioxidative system in 44 Korean alcoholics and 45 age-, sex-, and nationality-matched nonalcoholic subjects. Plasma triacylglycerols and atherogenic index were higher in alcoholics than in control subjects. Plasma total cholesterol was not different among groups, but plasma high-density lipoprotein cholesterol was lower in alcoholics. There were positive correlations between ethanol consumption and plasma lipid peroxide and atherogenic index in all subjects; there were negative correlations between ethanol consumption and plasma high-density lipoprotein cholesterol in all subjects. There were no significant differences between alcoholics and control subjects in plasma concentrations of α-tocopherol, although plasma α-tocopherol/lipid tended to be lower in alcoholics. Plasma retinol was lower in alcoholics. These results suggest that chronic ethanol consumption can contribute to increased risk for vascular diseases in Korean alcoholics.

Keywords: Alcoholics; Koreans; Lipid; Antioxidants; Retinol; α-Tocopherol

* Corresponding author. Tel.: +82 53 810 2875; fax: +82 53 815 2874.
E-mail address: jsseo@yu.ac.kr (J.S. Seo).

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1. Introduction

Recent data have shown beneficial effects of moderate drinking on the risk for cardiac disease [1]. A study in American Indian men and women showed a significant inverse association of alcohol consumption with peripheral arterial disease [2]. These studies, and others, have led to dietary recommendations that include moderate consumption of alcohol, especially in the form of red wine. Unfortunately, many people have consumed alcohol to excess. Alcohol intake was affected by both environmental and inherited biologic mechanisms [3]. Prolonged consumption of excessive amounts of alcohol increased medical risks for liver cirrhosis, several neuromuscular disorders, and several types of cancer [4]. The interaction of ethanol and lipid metabolism was relevant to the effect of alcohol consumption on the pathogenesis of alcoholic fatty liver and hyperlipidemia, and to atherosclerosis [5].

In the 20th century, the poor dietary habits of alcoholics were widely accepted as explaining several obvious connections between heavy drinking and organ damage. Ethanol consumption appeared to induce oxidative stress in the liver and in extrahepatic tissues [6]. Increased production of superoxide anion and hydrogen peroxide was derived from the oxidation of ethanol and its metabolites, leading to initiation of peroxidation [7].

Sensitivity to peroxidation was reported to be a function of the overall balance between pro-oxidants and antioxidants. Therefore, tissue antioxidants helped prevent the cellular damage caused by free radicals and free radical–mediated lipid peroxidation [8]. However, chronic alcohol consumption enhanced microsomal degradation of retinoids and promoted their depletion and associated pathology [9]. In addition, plasma concentrations of α-tocopherol and selenium were lower in alcoholics than in men who drank low amounts of alcohol, whereas malondialdehyde, a marker of oxidative stress, was higher [10].

Most studies of the nutrition of alcoholics and of the effects of alcohol on vitamin and antioxidant status have been conducted in the United States and Europe. People in these countries generally eat “Western diets” that are relatively fat-, energy-, and cholesterol-rich compared to the rest of the world. We hypothesized that the interaction between diet and alcohol, and the effects of alcoholism on nutrient status and metabolism, is influenced by the underlying diet of the alcoholic. Furthermore, alcoholics from different cultures drink to excess on different alcoholic beverages, on the basis of both the availability of the alcoholic beverage and on the dietary customs. The alcoholic beverage of choice might also influence their nutrient and oxidative damage status. Therefore, studies of the micronutrient and oxidative status of people eating non-Western diets, and drinking alcoholic beverages that are uncommon in the West, should provide important insights into the true relationships between alcohol consumption and nutrition. This human study was carried out to evaluate the effect of chronic alcohol consumption on plasma concentrations of lipid and antioxidative system in Korean alcoholics.

2. Methods and materials

2.1. Subject characteristics

The subjects were 44 Korean male chronic alcoholics (aged 30-50 years) hospitalized for a rehabilitation program and 45 Korean nonalcoholic males (aged 30-50 years). The control

subjects were recruited from healthy male workers of the same province. Subjects had no other chronic diseases and were not currently taking medications. We attempted to match the age distribution in the alcoholic group and the control group; mean ages were 43.0 ± 7.6 and 43.9 ± 7.5 years, respectively.

Participation in this study was voluntary and each subject gave their informed consent before donating blood. The study protocol followed the Helsinki Guidelines as revised in 1983.

A detailed standardized interview was carried out by trained interviewers with emphasis on the subject's drinking history, dietary habits, and drug intake. The alcoholic subjects were active drinkers with a history of alcohol consumption for more than 10 years. They consumed excessive quantities of alcohol until the day they entered the rehabilitation program and were hospitalized. Mean daily ethanol intake of the alcoholic subjects before entering the rehabilitation program was 132.6 g per day. Most of these alcoholics drank Korean hard liquor (Soju) more than 3 or 4 times a week before hospitalization. Soju is a distilled alcohol providing little nutritional value except energy, with an ethanol content of about 25%. The mean daily ethanol intake of control subjects was 3.2 g per day.

Fasting blood samples were collected from the antecubital vein of the alcoholic subjects early in the morning after their first night of hospitalization. Plasma was separated by centrifugation at 1400 × *g* for 15 minutes. Plasma samples were promptly stored at −80°C for analysis. Alcoholic subjects were provided with nutritional counseling and education for 1 month to promote their healthy rehabilitation.

2.2. Biochemical analyses

Serum concentrations of triacylglycerol, total cholesterol, and high-density lipoprotein cholesterol (HDL-C) were assayed enzymatically using commercial kits. Atherogenic index (AI) was calculated by an equation using plasma levels of total cholesterol and HDL-C [11]. All diagnostic kits were supplied by Youngdong Pharmaceutical Company, Korea.

Plasma concentrations of lipid peroxide were measured by a simple fluorometric assay [12]. The *n*-butanol layer of the thiobarbituric acid (TBA)–reacting mixture was collected for fluorometric measurements, which were made at 515-nm excitation and 553-nm emission. Fluorescence intensity of the standard solution was obtained by reacting tetraethoxypropane (Sigma) with TBA. Superoxide dismutase activity in plasma was determined by the method of Marklund and Marklund [13]. Enzyme activity inhibiting 50% of autoxidation of pyrogallol solution was defined as one unit of superoxide dismutase activity.

Simultaneous determinations of plasma retinol and tocopherol were by high-performance liquid chromatography using a modified method from Bieri et al [14]. Two hundred microliters of plasma was mixed with the same volume of retinyl acetate solution (80 μg/mL) and tocopheryl acetate solution (100 μg/mL) as internal standards. Blood lipid was thoroughly extracted with spectrograde hexane. The vials of hexane extracts were centrifuged to separate phases. The hexane extracts were filtered through a 0.45-μm nylon membrane into a small vial and evaporated under a stream of nitrogen. The dried extract was dissolved in chromatographic solvent (petroleum ether-methanol, 1:1 vol/vol). Twenty microliters of the reconstituted sample was injected onto the high-performance liquid chromatography (Waters 6000A, Milford, MA) equipped with C_{18} Bondapack cartridge column (30 cm × 3.9 mm, 10

Table 1
Plasma concentrations of triglyceride, total cholesterol, and HDL-C in alcoholics and control subjects

Group	TG	Total cholesterol	HDL-C	AI[a]
Controls (n = 45)				
mg/dL	92.0 ± 27.6[b,*]	191.6 ± 33.4**	45.7 ± 6.6*	4.31 ± 2.19*
μmol/L	1040 ± 312	4.96 ± 0.86	1.18 ± 0.17	
Alcoholics (n = 44)				
mg/dL	149.2 ± 25.2	218.4 ± 42.7	28.4 ± 4.6	7.02 ± 2.64
μmol/L	1686 ± 285	5.66 ± 1.10	0.74 ± 0.13	

Data are represented first in the familiar milligrams per deciliter format. Conversion factors for SI units are from http://www.unc.edu/~rowlett/units/scales/clinical_data.html. Conversion factors are mg/dL triacylglycerols = 11.3 μmol/L; mg/dL total and HDL cholesterol = 0.0259 μmol/L.
TG indicates triglyceride.
[a] AI = (total cholesterol − HDL-C)/HDL-C.
[b] Values shown are Mean ± S.D.
* Significantly different between controls and alcoholics at $P < .05$ by Student t test.
** Significantly different between controls and alcoholics at $P < .01$ by Student t test.

μm). The mobile phase was a mixture of methanol and water (95:5 vol/vol). Retinol and α-tocopherol were measured with UV detection at 280 nm.

2.3. Statistical methods

Results are presented as means and standard deviations. Group means were compared by Student t test after preliminary analysis of variance. Differences were considered statistically significant at $P < .05$. Pearson's correlation coefficients were determined between ethanol intake and biochemical parameters in all subjects. All statistical tests were performed using SPSS for Windows (version 10.0, SPSS, Chicago, Ill).

3. Results

Table 1 shows the plasma concentrations of triglyceride and cholesterol in alcoholics and control subjects. Triacylglycerol concentration in alcoholics was significantly higher than that of controls. Plasma total cholesterol showed no significant differences between alcoholics and controls. High-density lipoprotein cholesterol level of alcoholics was lower

Table 2
Pearson's correlation coefficients between alcohol intake and biochemical parameters in all subjects (n = 89)

Parameter	TBARS	TG	Total cholesterol	HDL-C	AI[a]
Alcohol intake (g/d)	0.67**	0.15	0.14	−0.29*	0.44**

TBARS indicates TBA-reacting substances.
[a] AI = (total cholesterol − HDL-C)/HDL-C.
* Significant at $P < .05$ by Pearson's correlation coefficients.
** Significant at $P < .01$ by Pearson's correlation coefficients.

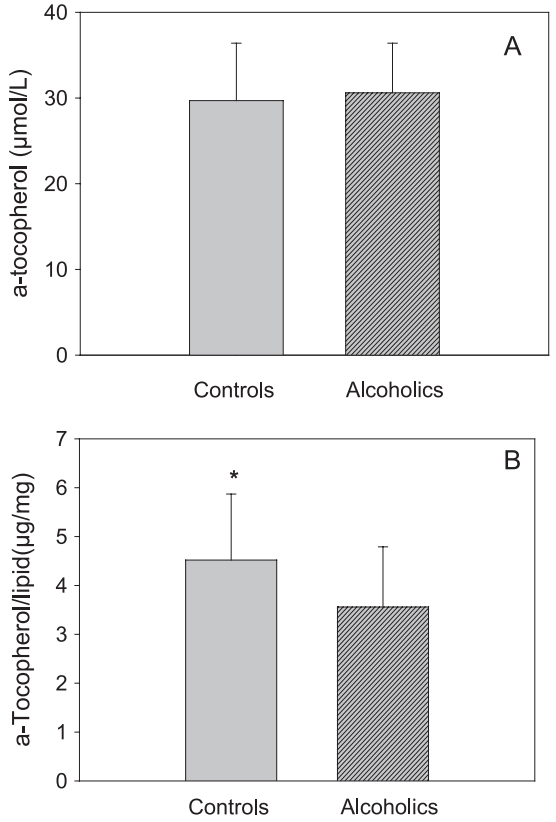

Fig. 1. Plasma levels of α-tocopherol (A) and α-tocopherol/lipid (B) in alcoholics and control subjects. Lipid: total triglyceride plus total cholesterol. Asterisk indicates significant differences between controls and alcoholics at $P < .05$ by Student t test.

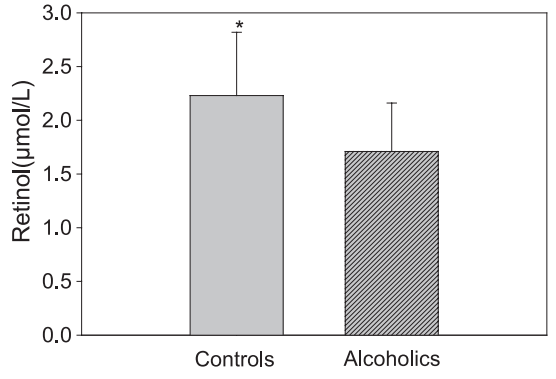

Fig. 2. Plasma levels of retinol in alcoholics and control subjects. Asterisk indicates significant differences between controls and alcoholics at $P < .05$ by Student t test.

Table 3
Plasma concentration of lipid peroxide and plasma superoxide dismutase activity of alcoholics and control subjects

Group	TBARS (μmol/L)	SOD (Unit/L)
Controls (n = 45)	4.2 ± 1.43[a,*]	3100 ± 580*
Alcoholics (n = 44)	4.5 ± 1.82	3400 ± 1900

SOD indicates superoxide dismutase.
[a] Values shown are mean ± SD.
* Significantly different between controls and alcoholics at $P < .05$ by Student t test.

than controls. The AI (calculated using total cholesterol and HDL-C levels) was higher in alcoholics as compared to the control subjects. Furthermore, we found a positive correlation between the AI ($r = 0.44$, $P < .01$) and ethanol consumption in all subjects, and a negative correlation between ethanol consumption and plasma HDL-C ($r = -0.29$, $P < .05$) in all subjects (Table 2).

Plasma level of α-tocopherol did not differ significantly between groups, although plasma α-tocopherol/lipid was lower in alcoholics (Fig. 1). Plasma concentrations of retinol are given in Fig. 2. Plasma retinol in alcoholics (1.71 ± 0.45 μmol/L) was lower than in control subjects (2.23 ± 0.59 μmol/L). When retinol concentrations were corrected for circulating lipid, this difference increased.

Plasma lipid peroxide and superoxide dismutase activity in alcoholics were not different from those of control subjects (Table 3). However, we found a positive correlation between ethanol consumption and plasma levels of lipid peroxide ($r = 0.67$, $P < .01$) in all of the subjects (Table 2).

4. Discussion

Alcohol abuse is a major cause of health problem and a public health issue [15]. Several studies have shown that the incidence of alcoholism and the mortality of patients with alcoholic liver disease have been increasing in Korea [16].

Hyperlipidemia associated with alcohol consumption is relevant to the problem of atherosclerosis and heart disease in the drinking population. Elevated serum triacylglycerols, low-density lipoprotein cholesterol (LDL-C), and decreased levels of HDL-C were shown to be risk factors for cardiovascular disease [17]. In this study, we found that plasma concentrations of triacylglycerol in Korean alcoholics were significantly higher than those in the control subjects. Also, the HDL-C level of alcoholics was lower than that of the controls, and the AI was higher in alcoholics. Korean people usually eat non-Western diets that contain less energy and fat compared to Western people. However, Korean alcoholics still showed high plasma concentration of triacylglycerols, especially compared to controls.

Population-based studies report a significant relationship between alcohol consumption and changes in blood lipids and lipoproteins; higher circulating triacylglycerol is a consistent finding in studies of individuals with high alcohol consumption, but is less consistent in subjects with ethanol intake below 60 to 80 g per day [18]. The interaction between ethanol and lipid metabolism is relevant to the effect of alcohol consumption on body weight and

body composition, to the pathogenesis of alcoholic fatty liver and hyperlipidemia, and atherosclerosis [19]. Pathogenesis of alcoholic fatty liver and hyperlipidemia was due mainly to a combination of decreased fatty acid oxidation in mitochondria and increased glycerolipid synthesis [20]. Siler et al [21] reported that even low-dose (24 g) ethanol consumption in healthy volunteers modestly activates hepatic de novo lipogenesis and that the major quantitative fate of ethanol is acetate produced in the liver. They also explained that the acetate released into the plasma inhibits lipolysis in peripheral tissues whereas whole body lipid oxidation is decreased.

However, some authors argued that the risk of developing liver disease does not increase over a threshold alcohol intake of about 75 g per day [22]. Furthermore, an inverse association between moderate alcohol consumption and coronary heart disease (CHD) has been observed in several epidemiological studies [23]. This reduction in risk of CHD could be a consequence of a decrease in circulating levels of LDL cholesterol [24]. However, another important change in serum lipids in moderate drinkers is an increase in HDL-C. At least half of the reduced risk in cardiovascular disease associated with moderate alcohol consumption was attributed to changes in circulating levels of HDL and HDL subfractions [25]. These reports suggest that alcohol taken in moderation may prevent atherosclerosis.

Heavy drinking appeared to have the opposite effect, in part by promoting oxidation of LDL, a pathogenetic factor in atherogenesis. Stivastava et al [18] in a review suggested that some of the inconsistency seen in the response of cholesterol to ethanol consumption might be related to the fat level in the diet. They suggested that individuals consuming a high-fat diet might be more sensitive to the effects of alcohol on blood lipids. According to the report of the Korean National Nutrition Survey, the average intake of fat of Korean adults was 19.0% of total energy [26], much lower than the 35% to 40% of energy typical in the West. Other aspects of the effect of alcohol drinking are still unresolved, namely, the effects of type of alcoholic beverage usually consumed, duration of drinking, sex, age at start, and time since quitting [17,27]. Our study in Korean alcoholics, who eat a non-Western diet and drink a different form of alcoholic beverage than studied in most reports, could be useful to determine some of the effects of the underlying diet and of the type of alcohol consumed. Our alcoholic group consumed dangerously high amounts of alcohol before their hospitalization, much more than the 158.9 g of alcohol per week used to define alcoholism by the National Institutes of Health [28]. However, our group consumed similar amounts as those consumed by early at-risk alcoholics [28], whose intake for men was 135 g of alcohol per day of drinking, or 630 g per week. Korean alcoholics tended to consume a diet that is lower in fat than their Western counterparts, and their beverage of choice is Soju, a fermented hard liquor that is uncommon in the West.

Alcohol-induced oxidative stress is linked to the metabolism of ethanol. Three metabolic pathways of ethanol have been described in the human body so far. Each of these pathways can produce free radicals that affect the antioxidant system [29]. Specifically, cytochrome P450 in the microsomal ethanol-oxidizing system can be induced by administration of a large ethanol dose or by chronic ethanol injection, has a high oxidase activity, and plays a crucial role in the microsomal generation of reactive oxygen species that have the capability of initiating membrane lipid peroxidation [30]. Sensitivity to peroxidation is a function of the overall balance between pro-oxidants and antioxidants. Free radicals increase the oxidative

modification of LDL. This is one of the most important mechanisms, which increases cardiovascular risk in chronic alcoholic patients. There was no significant difference in lipid peroxide level between alcoholics and control subjects in our study, but lipid peroxidation was correlated to ethanol intake in all subjects.

Dietary antioxidants play a role in preventing CHD by having an impact on lipid levels [31]. Antioxidant protection includes both enzymatic and nonenzymatic defense systems. Impairment of such systems has been reported in alcoholics. LeComte et al [10] reported that important enzymatic antioxidant systems—superoxide dismutase and glutathione peroxidase—were higher in alcoholics and significantly decreased after withdrawal. These changes could due to the direct effect of ethanol or to the ethanol-associated malnutrition [32]. The effect of heavy alcohol consumption on serum concentrations of α-tocopherol and selenium can be dramatic. Reduced serum levels of α-tocopherol and selenium may influence the maintenance of normal cell structure and function, and contribute to the development of diseases frequently observed in alcoholics [33]. Studies related to vitamin and antioxidant nutrition of alcoholics are very limited in Korea. Serum α-tocopherol in one study of Korean patients with alcoholic liver disease was decreased compared to controls, but vitamin A deficiency was not detected [34]. In our study, plasma superoxide dismutase activity and concentrations of α-tocopherol in alcoholics were not different from those of control subjects. However, when α-tocopherol concentrations were corrected for circulating lipid, it tended to decrease in alcoholics.

We found that plasma concentrations of retinol in alcoholics were decreased compared to control subjects. When retinol concentrations were corrected for circulating lipid, this difference increased. Although the diets of alcoholics may well be low in carotenoids and performed vitamin A, a direct effect of alcohol consumption on vitamin A metabolism has been indicated by the decrease in hepatic vitamin A seen in baboons and rats receiving nutritionally adequate diets containing alcohol [35]. Decreased plasma vitamin A in cirrhosis was reported to be partly attributable to decreased release of vitamin A from the liver because of decreased synthesis of retinol binding protein and prealbumin necessary for its transport [36]. However, Zima et al [29] did not find any changes of serum retinol and tocopherol concentrations in alcoholics.

Like ethanol, retinol is an alcohol and, in vitro, both can be converted to corresponding aldehydes in reactions catalyzed by several isozymes of cytosolic alcohol dehydrogenase. Substantial evidence indicated that one consequence of alcohol intoxication was a reduction in retinoic acid levels [37]. It is not surprising, therefore, that in vitro, and possibly in vivo, these 2 alcohols can interact significantly by competing with each other for the same or similar enzymatic pathways and by interfering with each other's reactions. Such interactions also involve the main retinol precursor, β-carotene.

In our study, chronic alcohol intake did not have a significant effect on lipid peroxidation and antioxidant system in alcoholics. However, chronic alcohol consumption appeared to contribute to lower HDL-C, higher triacylglycerol, and higher AIs, all of which may contribute to increased risk of vascular diseases. Further, vitamin A concentrations were lower in Korean alcoholics than controls, similar to older reports from the United States [36]. These results suggest that some of the effects of excess alcohol intake on nutrient intakes occur in people eating low fat as well as high fat diets.

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References

[1] Sillanaukee P, Koivula T, Jokela H, Pitkajarvi T, Seppa K. Alcohol consumption and its relation to lipid-based cardiovascular risk factors among middle-aged women: the role of HDL(3) cholesterol. Atherosclerosis 2000;152:503-10.
[2] Fabsitz RR, Sidawy AN, Go O, Lee ET, Welty TK, Devereux RB, et al. Prevalence of peripheral arterial disease and associated risk factors in American Indians: the Strong Heart Study. Am J Epidemiol 1999;149:330-8.
[3] Forsander OA. Dietary influences on alcohol intake: a review. J Stud Alcohol 1998;59:26-31.
[4] Halsted CH. Folate requirements and metabolism in alcoholic liver disease. Nutr Res 1994;14:1439-56.
[5] Lawrence F, Lieber CS. Ethanol and lipid metabolism. Am J Clin Nutr 1999;70:791-2.
[6] Nordmann R. Alcohol and antioxidant systems. Alcohol Alcohol 1994;29:513-22.
[7] Gasberrini A, Borle AB, Caraceni P, Colantoni A, Farghali H, Bernardi M, et al. Effect of ethanol on adenosine triphosphate, cytosolic free calcium and cell injury in rat hepatocytes: time course and effect of nutritional status. Dig Dis Sci 1996;41:2204-12.
[8] Henning SM, Zhang JZ, McKee RW, Swendseid ME, Jacob RA. Glutathione blood levels and other oxidant defense indices in men fed diets low in vitamin C. J Nutr 1991;121:1969-75.
[9] Leo MA, Lieber CS. New pathway for retinol metabolism in liver microsomes. J Biol Chem 1985;260:5228-31.
[10] LeComte E, Herbeth B, Pirollet P, Chancerelle Y, Arnaud J, Musse N, et al. Effect of alcohol consumption on blood antioxidant nutrients and oxidative stress indicators. Am J Clin Nutr 1994;60:255-61.
[11] Haglund O, Loustarinen R, Wallin R, Wibell I, Saldeen T. The effects of fish oil on triglycerides, cholesterol, fibrinogen and malondialdehyde in human supplemented with vitamin E. Eur J Nutr 1991;121:165-72.
[12] Yagi K. Short communications: a simple fluorometric assay for lipid peroxide in blood plasma. Biochem Med 1976;15:212-6.
[13] Marklund S, Marklund G. Involvement of the superoxide anion radical in the autoxidation of pyrogallol and a convenient assay for superoxide dismutase. Eur J Biochem 1974;47:469-74.
[14] Bieri JG, Tolliver JJ, Catignani GL. Simultaneous determination of tocopherol and retinol in plasma or red cell by high pressure liquid chromatography. J Clin Nutr 1979;32:2143-8.
[15] Choi EY, Im YS, Kim KN, Park CY, Kim HJ. Alcohol habits in Korea. J Korean Acad Fam Med 1998;19:858-69.
[16] Kweon YO, Kweon JG, Choi YH, Lee JN, Chung JM. Psychosocial characteristics of patients with alcoholic liver disease. Korean J Gastroenterol 1998;32:355-69.
[17] Manninen V, Elo MO, Frick MH, Haapa K, Heinonen OP, Heinsalmi P, et al. Lipid alterations and decline in the incidence of coronary heart disease in the Helsinki heart study. JAMA 1988;260:641-51.
[18] Stivastava LM, Vasisht S, Agrawal DP, Goedde HW. Relation between alcohol intake, lipoproteins and coronary heart disease: the interest continues. Alcohol Alcohol 1994;29:11-24.
[19] Liu S, Sedula MK, Williamson DF. A prospective study of alcohol intake and change in body weight among US adults. Am J Epidemiol 1994;140:912-20.
[20] Baraona E, Lieber CS. Alcohol and lipids. In: Galanter M, editor. Recent developments in alcoholism. New York: Plenum Publishing Corp; 1998. p. 97-134.
[21] Siler SQ, Neese RA, Hellerstein MK. De novo lipogenesis, lipid kinetics, and whole-body lipid balances in humans after acute alcohol consumption. Am J Clin Nutr 1999;70:928-36.
[22] Sorensen TIA. Alcohol and liver injury: dose-related or permissive effect? Liver 1989;9:189-97.

[23] Ajani UA, Gaziano JM, Lotufo PA, Liu S, Hennekens CH, Buring JE, et al. Alcohol consumption and risk of coronary heart disease by diabetes status. Circulation 2000;102:500-5.
[24] Clevidence BA, Reichman ME, Judd JT, Muesing R, Schatzkin A, Schaefer EJ, et al. Effects of alcohol consumption on lipoproteins of premenopausal women. A controlled diet study. Arterioscler Thromb Vasc Biol 1995;15:179-84.
[25] Langer RD, Criqui MH, Reed DM. Lipoproteins and blood pressure as biological pathways for effect of moderate alcohol consumption on coronary heart disease. Circulation 1992;85:910-5.
[26] Report on 1998 National Health and Nutrition Survey (Dietary intake survey). Seoul: 1999.
[27] Frezza M, Di Padova C, Pozzato G. High blood alcohol levels in women. The role of decreased gastric alcohol dehydrogenase activity and first-pass metabolism. N Engl J Med 1990;322:95-9.
[28] Enoch G. The Physicians' guide to helping patients with alcohol problem; 1995. p. 1-12 [NIH, Bethesda, MD publication No. 95-3769].
[29] Zima T, Fialova L, Mestek O, Janebova M, Crkovska J, Malbohan I, et al. Oxidative stress, metabolism of ethanol and alcohol-related diseases. J Biomed Sci 2001;8:59-70.
[30] Eckstrom G, Ingelman-Sundberg M. Rat liver microsomal NADPH-supported oxidase activity and lipid peroxidation dependent on ethanol-inducible cytochrome P 450(P450 llE1). Biochem Pharmacol 1989;38:1313-9.
[31] Slattery ML, Jacobs DR, Dyer A, Benson J, Hilner JE, Caan BJ. Dietary antioxidants and plasma lipids: the CARDIA Study. J Am Coll Nutr 1995;14:635-42.
[32] Addolorato G, Capristo E, Greco AV, Stefanini GF, Gasbarrini G. Energy expenditure, substrate oxidation and body composition in chronic alcoholism: new findings from metabolic assessment. Alcohol Clin Exp Res 1997;21:962-7.
[33] Bjorneboe GE, Johnsen J, Bjorneboe A, Bache-Wiig JE, Morland J, Drevon CA. Diminished serum concentration of vitamin E in alcoholics. Ann Nutr Metab 1988;32:56-61.
[34] Koo BK, Chung JM, Lee HS. Biochemical evaluation of nutritional status of vitamins and minerals in patients with alcoholic liver disease. J Korean Soc Food Sci Nutr 1998;27:1244-52.
[35] Sato M, Lieber CS. Hepatic vitamin A depletion after chronic ethanol consumption in baboons and rats. J Nutr 1981;111:2015-23.
[36] Smith FR, Goodman DS. The effect of diseases of the liver, thyroid and kidneys on the transport of vitamin A in human plasma. J Clin Invest 1971;51:2426-36.
[37] Molotkov A, Duester G. Retinol/ethanol drug interaction during acute alcohol intoxication in mice involves inhibition of retinol metabolism to retinoic acid by alcohol dehydrogenase. J Biol Chem 2002;277(25): 22553-7.